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The Pathophysiology, Presentation, and Triage of Altitude-Related Decompression Sickness Associated with Hypobaric Chamber Operation

DONALD C. ARTHUR and ROBERT A. MARGULIES

Naval Submarine Medical Research Laboratory, Groton, Connecticut

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Decompression sickness following excursions to low atmospheric pressures has recently been a topic of confusion and concern. This article provides a reference for management of decompression sickness occuring after exposure to a reduction in ambient pressure in a hypobaric chamber. The pathophysiology, recognition, classification, initial management, definitive treatment, and eventual disposition of these cases are presented in a form which is applicable to all flight surgeons and flight physiologists, especially those with responsibility for utilization of hypobaric chambers.

THE EFFECT OF HYPOBARIC chamber excursions on one's ambient pressure is analogous to SCUBA diving and deep-sea diving. Inherent, therefore, is the possibility of decompression sickness. This article summarizes the pathophysiology, classification, and a philosophy for treatment of altitude-related decompression sickness with some practical guidelines for safe hypobaric chamber operation.

Decompression sickness (DCS) is the result of a series of pathophysiologic responses to tissue gas evolution brought about by changes in ambient pressure. Bubbles, which are released from tissue equilibrium by a too-rapid reduction in ambient pressure, either obstruct blood flow, cause blood chemistry changes, or stretch and damage tissues. The symptoms can range from innocuous skin itching to serious central nervous system or pulmonary compromises. Prompt diagnosis and recompression treatment are essential.

Dr. Arthur is currently stationed at the Naval Air Station, Cubi Point, Philippines, and Dr. Margulies is currently teaching at the Uniformed Services University of the Health Sciences, Bethesda, MD.

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PATHOPHYSIOLOGY REVIEW

At steady state, the body tissues are normally equilibrated with gases contained in inspired air. The tissue partial pressures of these gases are primarily dependent on the ambient pressure, the concentration of gas within the breathing mixture, and the rate at which the gas is either removed or metabolized within the tissue. The rate of change in the partial pressures of the dissolved inert gases is proportional to the gradients between them and the ambient partial pressures (more correctly, the alveolar gas partial pressures); the greater the pressure gradient, the faster the uptake or elimination. A too-rapid reduction in ambient pressure may cause a gradient so great that the ability of the body tissues and blood to hold the gas is exceeded, resulting in bubble formation. An analogy can be drawn to the opening of a soda bottle too quickly, resulting in rapid liberation of carbon dioxide as bubbles.

A number of factors determine the amount of dissolved gases in the tissues and their rapidity of liberation. Obesity, exercise, age, and, possibly, sex are important subject factors (4,13,28,32). Exercise increases uptake and elimination by increasing the respiratory rate, cardiac output, and tissue perfusion, increasing the rate of gas delivery/removal at the tissue level (9,11,23, 24,26,32,41,58). Gas and body temperature and the state of the subject's hydration will proportionately affect the rates of uptake and elimination (8,30,55). Smoking and alcohol consumption will also increase susceptibility to DCS by a variety of mechanisms. Tissue injury, acute

or chronic, may increase susceptibility to DCS in the injured area (3,37,40,58). The rate of ascent and the time at altitude are the most important factors in determining the rate of elimination and, hence, the likelihood of bubble formation (22,25,41,50,58).

Body tissues can be categorized by tissue half-times: the time required to become one-half saturated with a gas in response to a given increase in ambient pressure. Muscle, for example, has a very short half-time; bone and fat have very long half times. Rates of gas elimination are proportional to rates of gas uptake but probably take much longer during recompression. A tissue such as fat can take on a large amount of nitrogen since nitrogen is five times more soluble in fat than in water (33,35). This tissue, however, eliminates nitrogen very slowly and supersaturation with subsequent bubble formation can easily occur. Thus, obese people seem to experience DCS more readily than lean people (3,10,13,28,44,58). In diving, this is usually true only after deep and very long dives when the slow adipose tissues can accumulate enough gas for supersaturation to occur. In altitude-related DCS, however, the adipose tissue is initially saturated, possibly predisposing obese subjects to increased bubble formation within the adipose tissues. Although bubbles confined to fatty tissues do not often result in symptoms, their systemic dissemination could.

Unlike nitrogen and other inert gases, oxygen is rapidly utilized in the normal metabolism of every cell and is, therefore, rapidly removed from solution within the tissues, decreasing its partial pressure. Breathing 100% oxygen eliminates the supply of inert gas to the tissues, thereby creating a partial pressure gradient favorable to elimination of inert gas (21,59). This principle is applied to aviators who use 100% oxygen from take-off to landing, thereby decreasing the tissue tension of nitrogen, the major constituent of air, and, hence, of the bubbles of DCS. (Granted, this is not the primary reason for oxygen use in aviation.) This "washout" principle is also utilized when applying 100% oxygen to victims of DCS—providing conditions favorable to bubble dissolution while providing an enriched supply of oxygen to ischemic tissues.

Bubble formation resulting from the combination of ascent rate and time at altitude (usually the only variables) is the *sine qua non* of DCS. The reduction in pressure can result from either an ascent from ground level to a high altitude, as in a hypobaric chamber, or by surfacing from a dive, where each 33 feet of sea water depth adds 1 atmosphere to the ambient pressure. The symptoms caused by bubbling are dependent on the location rather than merely the presence of the bubbles. Doppler studies have demonstrated that not all divers who have intravascular bubbles exhibit symptoms of DCS (1,6,42,49,51). Symptoms tend to develop when the bubbles are either in a strategic location, when the lungs cannot eliminate them by rapid diffusion into the alveoli, or when the blood chemistry is significantly altered. Small bubbles, located within tendons or other nonexpandable tissues, may cause pain as nerve endings are stretched, resulting in the classic "bends" or joint pain. Overloading the pulmonary vasculature with bubbles may result in a symptom complex known as "chokes," and intravascular bubbles (usually venous) within the central nervous system may result in CNS symptoms.

Differentiation must be made between evolved gas, trapped gas, and gas liberated within tissue planes. EVOLVED GAS refers to bubbles within tissues or body fluids which are liberated from solution in body tissues. DCS is defined as a manifestation only of evolved gas; thus a synonym for DCS is "evolved gas dysbarism." Symptoms will occur on or after ascent and may become apparent after return to ground level. These symptoms may not be relieved by merely returning to the original ambient pressure; additional increases in ambient pressure, as in a hyperbaric (recompression) chamber are usually required (10,14,21,50,54). A delay in appearance of symptoms may be due to the time required for coalescence of minute bubbles and for mobilization of body defenses, which may further add to the insult as described below in the discussion of Type II DCS.

TRAPPED GAS dysbarism is caused by the expansion of preexisting gas within a body cavity, such as the middle ear, sinuses, or gastrointestinal tract. Thus, trapped gas effects will occur *during ascent* as the trapped gases expand and cause pain, and will be relieved by returning to the original ambient pressure. This pattern of symptom onset is in marked contrast to that of evolved gas effects, where symptoms occur during and after exposure to the lower ambient pressure and will usually persist after return to the original ambient pressure. Proper treatment requires differentiation between symptoms due to evolved gas and those due to trapped gas. The history of the altitude or diving profile and time of onset of symptoms is extremely helpful.

Likewise, gas which is liberated BETWEEN TISSUE PLANES from a ruptured alveolus is not a manifestation of DCS. Mediastinal and/or subcutaneous emphysema and pneumothorax are examples. A ruptured alveolus can also lead to introduction of gas bubbles directly into the pulmonary vasculature and, subsequently, the systemic circulation leading to the disastrous consequences of gas embolism.

It is extremely important to differentiate these symptoms from those of DCS to institute proper treatment. For example, recompression therapy is contraindicated in pneumothorax (unless DCS is present concomitantly and a chest tube can be inserted) but is necessary immediately for arterial gas embolism. Mediastinal emphysema can also cause symptoms closely resembling chokes, and recompression is life-saving in the latter, but unnecessary in the former. The pressure profile and history of the onset of symptoms are extremely helpful, if not crucial, for such differentiation.

CLASSIFICATION OF DCS

Decompression sickness (evolved gas dysbarism) can be divided into three categories of varying severity: skin bends, Type I DCS and Type II DCS. The treatment of each is different and differentiation is mandatory. Skin bends is a manifestation of bubble formation within the skin and, when not associated with systemic symptoms, is not serious. Many authorities classify skin bends with Type I DCS, and treat them identically, reasoning that any manifestation of bubble formation may serve

as a harbinger of systemic bubble formation not yet clinically evident. Marbling of the skin is said to represent intradermal bubble formation; it should be considered indicative of impending systemic involvement, categorized with Type II DCS, and treated accordingly (7,14,15,18,25,38,50). Skin bends most commonly presents as itching alone, but may include rash formation and progress to marbling. Itching must be differentiated from the tingling of paresthesias or hypesthesias (described below), which are serious symptoms. The itching of skin bends is somewhat relieved by application of direct pressure to the area, whereas direct pressure will have no effect or will aggravate paresthesias. Skin bends is usually localized, but may involve a large area such as an entire extremity or the torso, but will not follow dermatome distributions.

Type I DCS describes the classic entity of "pain only" bends. Specifically, Type I DCS includes pain confined to the legs or arms. This pain may be aggravated by movement and may be relieved by direct pressure on the area, e.g. with a sphygmomanometer cuff (18,38,41,50). The pathophysiology probably consists of small bubbles within the tendons, etc., which cause pain by mechanical distortion (29,33-35,38,50). These can be no association with systemic symptoms for classification as Type I DCS. Once again, caution must be exercised to differentiate this pain from paresthesias, hypesthesias, or referred pain from central sites.

Type II DCS includes all other manifestations of evolved gas pathology. These are 1) pain elsewhere than cited above, 2) any CNS symptom, and 3) pulmonary manifestations (chokes). Pain in the head, neck, or torso is classified as a serious symptom. Although isolated hip and shoulder pain may not be a serious symptom, the pain may represent referred pain from visceral sites. Therefore, when hip and shoulder pain cannot be determined to be of musculoskeletal origin, inclusion as a Type II DCS symptom will assure proper treatment. Caution must be exercised in excluding the diagnosis of Type II DCS in favor of a "trapped gas syndrome" (barotrauma) manifesting as abdominal, sinus, or ear pain. Obstruction of the blood supply to some part of the central nervous system can become apparent as a deficit in any component of CNS function (12,17,36,53). The spinal cord is involved frequently, resulting in paresthesias along dermatomes or any of a variety of motor deficits ranging from segmental diminution in strength to quadraplegia. Intracranial pathology can impair any of the cranial nerves or cause regional deficits identical to the symptom pattern of a stroke, including seizure activity and cardio-respiratory collapse. CNS symptoms can also manifest themselves qualitatively as changes in the Mental Status Examination, such as subtle changes in mood, unwarranted fatigue, memory deficits, illusions, hallucinations, etc. Additionally, the inner ear can be involved yeilding vestibular (the "staggers") and auditory symptoms. High spinal cord and intracranial involvement are most common in aviation-related DCS, whereas lower spinal cord "hits", most frequently presenting as paraparesis, are more frequent in diving (14,15,21,50). Chokes is a manifestation of DCS resulting from the accumulation of bubbles in the pulmonary vasculature, which usually presents as a triad of cough, substernal pain, and dyspnea (9,25,38).

The pathology of DCS consists not only of the direct mechanical effects of bubble formation and migration, but also their indirect effects on blood elements, vascular tone, and vessel wall permeability (19,30,45,46,57). Platelets and plasma proteins coat the intruding bubble and increase local coagulation. Once platelet and local vessel damage has occurred, vessel occlusion is aggravated by platelet clumping, fibrin deposition, and activation of vasoactive agents. Perivascular damage is compounded by the local inflammatory response to injury. Hence, it is extremely important to treat victims of DCS as rapidly as possible to prevent irreversible local changes which make recovery and rehabilitation difficult. Platelet and coagulation changes occur within minutes; the inflammatory response is delayed, requiring hours to develop.

Adequate treatment necessitates prompt diagnosis and differentiation of DCS from less serious pathology. Caution must be exercised when the diagnosis of skin bends or Type I DCS is made, since their presence may foreshadow the development of Type II DCS symptoms (3,7,14-16,18,25,38,50). Symptoms can develop on ascent but are, more often, delayed as bubbles coalesce and the indirect effects accumulate. Thus, all participants in chamber runs should be observed for at least 1 h after return to site level. The flow chart of Fig. 1 is designed to facilitate initial assessment and management.

INITIAL MANAGEMENT

The nature of DCS pathology mandates recompression therapy to shrink or eliminate the offending bubbles. If bubbles obstruct cerebral or coronary blood flow, for example, CPR and other life-saving procedures will be ineffective without recompression. The function of initial management is stabilization for immediate medevac to a hyperbaric facility. Table I lists the suggested supplies for the on-site medical locker.

Provisions for prompt initial management and triage must be arranged prior to chamber operations. Participants must hold a current Clearance Notice ("up chit") and not have been SCUBA diving within the past 24 h (5,20,54). Exposures to above FL200 should not be repeated within 48 h and those to below FL200 within 24 h owing to the reported increased incidence of DCS in these groups (18,20,38,39,43).

Suspicion of DCS (a "hit") mandates immediate descent to site level as rapidly as practical, simultaneous activation of prearranged medical attention, and evacuation to a definitive hyperbaric treatment center. A flight surgeon should be no more than 5 min from the chamber during and for at least 1 h after the run to allow immediate diagnosis and triage.

The initial treatment for Type I and Type II DCS (skin bends without marbling or association with systemic symptoms does not require treatment) consists of 100% oxygen and rest in the left lateral Trendelenberg position (in an attempt to prevent any venous bubbles from reaching the left side of the heart and, subsequently, the arterial circulation). Type II DCS associated with any deterioration in vital signs should be the only indication for appropriate use of IV's, NG tubes, intu-

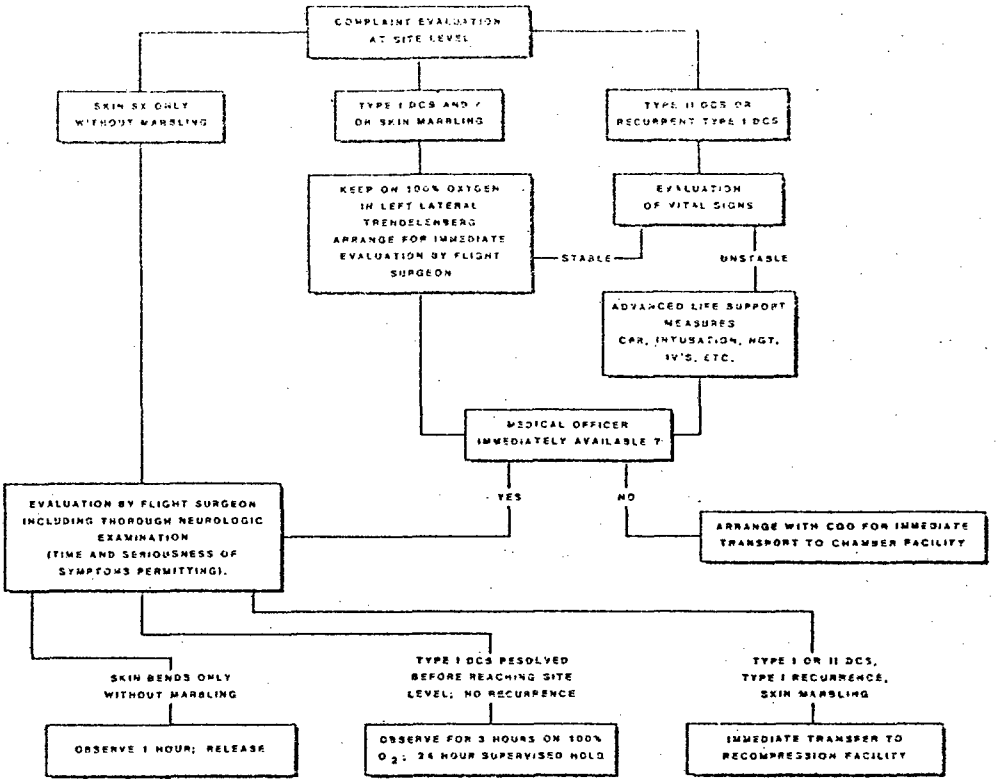

Fig. 1

bation, or CPR. If endotracheal intubation is performed, the cuff should be filled with saline to maintain a proper seal and prevent tracheal damage during recompression treatment. X-rays, blood sampling, filling out of forms, etc., waste precious time and will be attended to during or after recompression. Once the diagnosis of DCS has been made, the remainder of the physical examination should be performed enroute to the treatment facility.

The call for the flight surgeon should be followed by calls for medevac transportation and notification of the recompression facility. The patient should be transported as rapidly as possible even if unstable, continuing lift-

TABLE I. CONTENTS OF THE MEDICAL LOCKER.

DIAGNOSTICS:	flashlight, otoscope/ophthalmoscope, stethoscope, sphygmomanometer, reflex hammer, tuning fork, pin and brush sensory tester, tongue depressor.
AIRWAY KIT:	laryngoscope with extra batteries and bulbs, laryngoscope blades (McIntosh #3 & #4, Miller #2 & #3), suction catheters, cuffed endotracheal tubes with adapters (7.0, 8.0, 9.0), malleable stylet, oral airways (#3, 4, 5), nasal airways (32F, 34F), sterile lubricant, ambu bag with water-filled mask, oxygen cylinder and regulator with appropriate tubing, Magill forceps.
PNEUMOTHORAX KIT:	2-ml syringes with 21-ga needles, 2-ml of 2% lidocaine, disposable scalpel, 12- or 14-ga needles, Argyle trocar chest tubes, Heimlich one-way valves (or Penrose drains), McSwain dart, 2-0 suture material on cutting needle, suture holder, scissors, disposable sterile gloves.
*PHARMACEUTICALS:	Ringers lactate and normal saline (1000-ml bags, not bottles), atropine for injuction, bicarbonate for injection, calcium chloride for injection, lidocaide for injection, diazepam for injection, phenobarbital for injection, aminophylline for injection, dexamethasone for injection, sterile water for injection, dopamine.
MISCELLANEOUS:	padded tongue depressors, suction apparatus with hard-tipped catheters, waterproof adhesive tape (1- and 2-in), scissors, nasogastric tubes with extension tubing, IV infusion sets with IV extracaths (16- and 18-ga).

* Caution must be exercised in administration of pharmaceuticals; their effects will be diminished by the overriding pathology of the severe DCS which would require them. Their full effects will become apparent cumulatively when recompression reverses the underlying pathology.

support measures enroute. If transportation must be by air, cabin pressure should be kept as close to 1 atmosphere as possible (48). Primary and alternate recompression facilities should be identified, their daily status ascertained before chamber operations begin, and drivers should be familiar with the best routes to these facilities. Prior liaison with the recompression facility will ensure a smooth evolution.

If a physician familiar with DCS is not IMMEDIATELY available, arrangements should be made for immediate transport to a recompression facility if the experienced flight physiologist suspects DCS. Diagnosis of DCS does not *require* consultation with a physician.

DEFINITIVE TREATMENT—(5,6,10,12,14,21,33,40,48,50,52,54,59)

Skin bends not associated with other symptoms of DCS is generally considered innocuous and usually resolves without therapeutic intervention. Since one must be alert for the appearance of more serious symptoms of bubble formation, however, the patient with uncomplicated skin bends should be monitored for 1 h after the appearance of symptoms before being released. Although the occurrence of isolated skin bends should not affect the patient's flight status, the health record should reflect the incidence of any pressure-related medical problem.

Type I (pain only) DCS which resolves before reaching site level can be considered treated by the pressure increase inherent in the descent only if unassociated with more serious symptoms and without recurrence at site level. This victim should be directly observed for 3 h and supervised (accompanied, not hospitalized) for 24 h after return to site level. Type I DCS which initially resolves before return to site level but recurs is automatically classified as serious and is treated identically to Type II DCS.

Uncomplicated Type I DCS which is evident prior to return to site level and persists at site level or which initially manifests after return to site level is automatically treated on a Standard U. S. Navy Treatment Table 5. Type II DCS and recurrent Type I DCS are treated on a Standard U. S. Navy Treatment Table 6. Skin bends associated with marbling would be treated as if it were a Type II hit. The initial management and diagnosis from the hypobaric chamber facility may be modified by the treatment providers at the recompression facility as dictated by the standards for care of DCS outlined in the U. S. Navy Diving Manual (56), the ultimate source of guidelines in the hyperbaric treatment of DCS.

The usual practice after treatment is to admit the patient to the hospital for 24 h of observation. If a hospital is not nearby, the patient should remain in the immediate area of the recompression facility for the same 24-h period of observation. A Grounding Notice ("down chit") should be issued pending review of the physical qualifications of those receiving recompression treatment and for those with Type I DCS which resolves before reaching site level without recurrence. A thorough physical examination by a flight surgeon should precede issuance of an "up chit." The patient should be grounded for 1 week after a Type I episode and for 30 d following a Type II hit. Repeated incidents of Type I DCS do not preclude reexposure, but a second Type II DCS episode should warrant evaluation of the subject's fitness to undergo further exposures to hypobaric conditions. Specifically, predisposing factors should be investigated.

Decompression sickness is a serious illness for which there is only one safe and efficacious treatment. Suspicion of DCS should mandate recompression. Since the signs and symptoms of decompression sickness are often subtle and confusing, any questions involving diagnosis or initial management should be promptly referred to the diving physician at the hyperbaric facility. In addition, 24-h consultation is also available from the Experimental Diving Unit in Panama City, FL, at (904) 234-4351 (AUTOVON 436-4351) and through Duke University's Diving Accident Network at (919) 684-8111.

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